Correspondence

The Editorial Board will be pleased to receive and consider for publication correspondence containing information of interest to physicians or commenting on issues of the day. Letters ordinarily should not exceed 600 words, and must be typewritten, double-spaced and submitted in duplicate (the original typescript and one copy). Authors will be given an opportunity to review any substantial editing or abridgment before publication.

More HAFE

TO THE EDITOR: Drs. Auerbach and Miller are to be commended on their skilled and careful observations of the syndrome HAFE (high altitude flatus expulsion) reported in the February issue.[1] However, I would like to propose another etiologic mechanism.

I, too, have observed this phenomenon at similar altitudes and noted a predilection for its occurrence during the night and upon awakening in the morning, accompanied by borborygmus. It is known that arterial oxygen saturation drops to 88 percent in normal persons sleeping at 10,000 feet.[2] Acute decreases in arterial oxygen saturation at altitude may in turn lead to intestinal hypermobility and possible abnormalities of normal enzymatic and digestive function. Hyperactive bowels combined with other factors such as freeze-dried foods (high fiber content) and decreased barometric pressure (less than 500 mm of mercury at 11,000 feet) may provide a more fruitful explanation for this distressing syndrome.

EDWARD GEEHR, MD
Research Fellow
Department of Emergency Medicine
UCLA Hospital and Clinics
Los Angeles

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Gastrointestinal Barotrauma

TO THE EDITOR: In their exposé of the high altitude flatus expulsion (HAFE) syndrome, Auerbach and Miller[1] compared this entity to the development of decompression sickness in deep-sea divers. Actually, though, it would have been more appropriate to compare HAFE with the gastrointestinal barotrauma that occasionally occurs in divers because HAFE has exactly the same pathophysiology as this type of barotrauma.

Gastrointestinal barotrauma—which is also known as bowel barotrauma, aerogastralgia and gas in the gut—is caused by expansion of intraluminal bowel gas as ambient atmospheric pressure is decreased during ascent from a dive. It is manifested by eructation, flatus expulsion, abdominal fullness and colicky pain. Although it is rarely severe, it has been known to cause syncopal and shock-like states.[2]

Bowel barotrauma is seen more often in scuba divers than in deep-sea divers, who typically wear a full facemask or helmet, because scuba divers, especially if inexperienced, are more prone to swallowing air. Performing the Valsalva maneuver to clear the ears while in the head-down position during descent also forces air into the stomach. Other predisposing factors include drinking carbonated beverages or eating a heavy meal, particularly one containing legumes or other flatogenic substances, soon before diving, or chewing gum during the dive.

The only treatment that is usually needed for bowel barotrauma is slowing the rate of ascent or stopping ascent entirely until the excess bowel gas is vented. Sometimes, descent to a higher pressure is needed. Recompression is needed in only the most extreme cases.

Along this latter line, it is also relevant to note that hyperbaric oxygen therapy (HBOT) has been successfully used to treat some similar problems based on the converse principle that entrapped gas will decrease in size when compressed to elevated atmospheric pressure. Loder reported using HBOT to treat 12 cases of paralytic ileus which had failed conventional management.[3] Similarly, Kulak and co-workers reported using hyperbaric therapy to reduce the size of several intestinal catheter balloons and, thus, allow their removal when other methods of deflation had failed.[4] Also, HBOT has been used to treat pneumatosis cystoides intestinalis.[5]

Although HAFE and gastrointestinal barotrauma are of unlikely concern to most clinicians, it is of practical worth to remember that HBOT may be

potentially useful in treating various trapped gas syndromes that do not respond to conventional methods.

KENNETH W. KIZER, MD, MPH
Consultant in Diving and Hyperbaric Medicine
University of California, San Francisco Medical Center
San Francisco
President-Elect
North Pacific Chapter, Undersea Medical Society, Inc.

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Alkalinization Therapy for Tricyclic Antidepressant Overdose

TO THE EDITOR: I appreciated the insight into treatment of cardiac arrhythmias caused by tricyclic antidepressant overdose provided by Hoffman and McElroy in their recent article.[1] However, I take issue with the recommendation that "alkalinization therapy" be used to treat quinidine overdose. One of the authors' own references[2] actually states that quinidine urinary excretion is inversely related to urine pH, and that QT lengthening, a quinidine-induced effect, increases as urine pH increases. This suggests that urine pH exerts a clinically significant effect.

Two other references cited by Hoffman and McElroy[3,4] mention administration of alkalinizing drugs during quinidine toxicity, but only in combating the patients' preexisting metabolic acidosis. In neither case was *alkalinization* considered the major thrust of treatment, and both articles mention that although this procedure has been advocated, it has not always proved beneficial.

A case which illustrates the potential danger of an alkaline urine pH concomitant with quinidine administration was reported by Zinn in 1970.[5] In a patient whose condition was stabilized with constant doses of quinidine and digoxin, electrocardiographic signs of quinidine toxicity began to occur. A quinidine level subsequently obtained was 25 μg per ml. When questioned, the patient was found to have begun recently making his urine alkaline with his diet and antacid tablets. Upon return to a normal diet, quinidine serum levels returned to the therapeutic range at his previous dose.

The administration of sodium bicarbonate may be necessary in treating quinidine toxicity if metabolic acidosis has occurred. In this case it would be used in conjunction with specific measures to combat quinidine's ill effects (hypotension, convulsions, arrhythmia). Sodium bicarbonate should not be advocated as one of these specific measures.

MARY LINDA H. STOTTER, PharmD
Methodist Hospital of Indiana
Indianapolis

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TO THE EDITOR: I read with interest Hoffman and McElroy's excellent article on bicarbonate therapy in tricyclic antidepressant overdose in the January 1981 issue.[1] I was glad that they have thereby focused attention on this somewhat neglected and quite effective treatment. It thus pains me to nitpick an excellent presentation, but their discussion of phenytoin deserves some amplification.

First, an error apparently occurred in preparation of the manuscript, and the reference cited[2] regarding the ineffectiveness of phenytoin (diphenylhydantoin) contains no mention of that drug whatsoever. There is, in fact, scant literature on this topic. Until recently, there was only one published report recommending phenytoin in humans, and this did not mention the number of cases or the results. The authors suggested a dose of either 200 mg given intramuscularly or 5 to 10 mg per kg of body weight intravenously as a useful prophylactic agent against "cardiac or cerebral arrhythmias."[3] Also until recently, the only other published data involved three dogs given amitryptyline until arrhythmias developed. Intravenously given phenytoin in varying doses had a brief antiarrhythmic effect at that time, but sinus rhythm was not restored.[4] At the same time, pulse and blood pressure fell significantly and respirations were depressed, even in the one animal receiving only 2 mg per kg of body weight. The two animals that received 15 to 17 mg of phenytoin per kg of body weight died several hours later.

Fortunately, the most detailed report yet made